the removal of the primary disease, when that is possible, and to improving the general nutrition.

Alfred L. Loomis.

HEART: DIFFERENTIAL DIAGNOSIS OF ORGANIC VALVULAR DISEASES. I. As it is difficult to retain at all times in the mind the structure, complex operations and functions of the several chambers and valves of the heart, and the nature and cause of the modifications of the sounds they give forth when diseased, we shall therefore, whilst endeavoring to present a clear and simple exposition of the subject, make no apology to the readers of the HANDBOOK for the statement or repetition of an occasional truism, or of a very elementary and well-known fact.

The heart is a machine of exquisite construction, with four chambers and four sets of valves, its cavities incessantly flooded and emptied, and whilst supplying the motive power to renovate itself as well as the entire body it sometimes works for a century without repair; yet its mechanism and functions as a great receiving and driving engine—in its healthy action as well as for all purposes of the pathologist—*can* be grasped and understood by the mental eye if close attention be given to the subject. A knowledge of both its mechanism and functions must be attained as a preliminary step, by any one who pretends to pronounce upon its pathological changes; but so complex are the elements involved—diverse and diametrically opposite operations having to be appreciated and retained at the same time in the mind—that even when thoroughly comprehended, few are enabled to apply their knowledge practically at the bedside.

Notwithstanding, we will endeavor to evoke order out of this apparent confusion and difficulty, by means of a generalization which is of great practical use, and which we arrived at many years since, whilst teaching clinical medicine to successive classes. This is embodied in a very brief table or formula, which is characterized by exceeding simplicity, yet it is competent to the unravelling or interpretation of every uncomplicated case of valvular disease—for its merits are intrinsic, being based upon the relations existing between the mechanism and functions of the organ. The table, which will be developed in the course of this paper, is of instantaneous applicability, and can be used by anyone.[1]

No allusions will be made in this paper to hypertrophy, dilatation, pericarditis, or other diseases of the organ involving its muscular walls, external coverings, or its relations to the surrounding viscera.

It is almost needless to say that the first effort of one who is desirous of knowing whether the valves and orifices of the heart are diseased is, obviously, to notice if there be any *derangement*, *aberration*, or *change* from the *normal* sounds. He takes care to listen at the *base* and at the *apex*, paying *separate* attention to each point respectively; and also to the condition of the right and the left cavities,—in order, if he does discover any *morbid* sounds (a modification of the natural being always a morbid sound), that he may isolate and designate the derangement or lesion which such morbid sound surely indicates. It simplifies the process very much to know that far the greater number of endocardial lesions or injuries (it is needless to give the figures) are found in the *left* cavities. He should keep in mind also that the structure of the valves, or curtains at the *base* of the heart (the semilunar or sigmoid valves of the aorta and pulmonary artery) are analogous in shape, and act similarly and simultaneously. They are placed at their respective gateways with similar intent; they close and open, give ingress and egress to the column of blood synchronously. The same is true of those at the point or *apex* (the bicuspid or mitral, and the tricuspid). These, placed between the left and right auricles and ventricles respectively, differ essentially in form and structure from those at the base of the organ,—but they resemble *each other* in their general shape; *they* also open and close simultaneously, and perform analogous functions with each other in the economy of the organ. So, in making a diagnosis in the case of a heart supposed to be diseased, we address our examination to, and fix our mind upon, these *two sets* of valves separately, to see if any of them are diseased, and if so, to note: both what is the nature of the change which exists in their own structure, and what modifications have been produced by their alteration of form upon the *orifices* which they close and open. This essential method of procedure (coupled with the statement made above regarding the very marked *infrequency* of diseases of the right cavities), already greatly simplifies the study of the diseased valves. It is practised even by the novice in such inquiries; and when one is seen examining the heart at random—regardless of the above rule, it is clear to the looker-on that he has not mastered the first horn-book lesson upon the subject, and that it is impossible for him to form any accurate conclusions. He may know that the organ is diseased, but he cannot tell where the injury is.

Besides this, whoever is desirous of investigating a case of heart disease, must have in addition to his anatomical knowledge, fully and clearly in his mind the whole action and reaction in the cavities of the organ during its systole and diastole; he must know when and where the current is flowing out, and when and where its passage is estopped—whether at the back gates, or at the front gates, and conversely. For it is when those muscular and tendinous strings and cords at the apex, or those semilunar curtains at the base—which open and shut those orifices—are defective, *i.e.*, where they close imperfectly ("*insufficiency*"), and permit regurgitation when they should not; or when by fibrinous or other deposits upon the valves the orifices are *narrowed* or *roughened* ("stenosis"), and thus *obstruct* the forward flow, and give rise to abnormal, morbid sounds—it is the consideration and explanation of this problem which is his object in every case which becomes the subject of critical inquiry.

It is essential then, that besides the full appreciation of the currents and checks in the incessant working of the organ—the onward flow and the movements of the fleshly barriers which suddenly and rhythmically arrest the flow,—he should first *know* the normal healthy sounds, in order to detect the slightest *deviation* from them; and he should localize these deviations—for they are necessarily *morbid* sounds, and indicate *diseased valves*.

It being necessary then for the observer to know the cause and *rationale* of the normal sounds, we will state them. It is pretty well agreed that the first sound (represented by the word "lubb") is synchronous with the *systole* of the organ, and is owing to one or all of three causes: viz., the contraction of the muscular ventricles, the sudden closure of the auriculo-ventricular valves which prevents the blood from regurgitating into either auricle, and the impulsion of the heart against the walls of the chest. At this moment a column of blood is driven through the aorta and pulmonary artery, and the auricles are silently filling with blood from the valveless venæ cavæ and from the pulmonary veins.

That the second sound (represented by the word "dup") is synchronous with the *diastole* of the organ, and is due to the shutting up of the semilunar valves of the aorta and pulmonary artery. The closure of these valves at this moment prevents the regurgitation of the blood from the aorta and pulmonary artery into the ventricles—when during the diastole of the ventricles these are being filled from the auricles.

During the prolonged interval of rest following (which is equal in duration to the first, and twice the length of the second sound), we may suppose that the auricles are still silently pouring their contents into the ventricles—the portals of which are now wide open. During this period of apparent calm the heart—endowed with a high degree of nervous energy derived from the cardiac ganglia of the sympathetic and the pneumogastric, wound round and enwrapped with concentrically interlaced muscular fibres, layer upon layer as if encased with triple steel, and indeed the very "cunningest pattern of excelling nature" as respects endurance, strength and force—is preparing, like a wild animal gathering for its spring, for the next systolic paroxysm when its contents will be forced into the delicate meshes of the lungs, and be driven through

the finest capillary tubes in the remotest tissues of the organism.

We will confine our attention at present, whilst attempting to describe the *morbid* sounds and the lesions they indicate, to what takes place in the left cavities,—for whatever is true of the left is true of the right so far as the circulation of blood is concerned—and we shall simplify matters much by so doing.

Now with the first sound (systolic) the blood is being driven through the opened aortic orifice—at which moment the back-gate (the mitral or bicuspid) is shut. So, if we have a deranged or abnormal *first sound* heard with the greatest intensity at the *base* of the heart (and it is not a soft, inorganic, anæmic murmur, which is owing to the thinness of the blood, and which is out of the present question), there is necessarily a narrowing (stenosis) or roughness of the aortic orifice,—an obstruction there by vegetations, atheroma, or other morbid condition preventing the natural flow of blood through the aortic orifice, and deranging or modifying the natural sound.

Hence a deranged first sound at the *base* of the heart indicates *narrowing* or obstruction at the orifice—stenosis, in other words, of the *aortic valves*.

But suppose this abnormal, *first sound* has its greatest intensity at the *apex* of the heart. It must be owing to this fact: that the back-gate has a chink in it—it is more or less open, in place of being tightly closed as it should be; the column of blood, instead of meeting with the normal resistance of the closed and perfect mitral valve (bicuspid) in order that it may be propelled through the aorta and reach the utmost boundaries of the tree of life—is leaking back through the defective portals of the mitral,—it is regurgitating into the left auricle; and it gives out to the ear placed over the apex a morbid murmur or noise, more or less *prolonged*, in place of the ordinary normal first sound (represented by the word "lubb"). The valve is necessarily defective as a flood-gate; it is incapable of close shutting up; that its mechanism has become defective is indisputable, and we pronounce positively upon the subject.

So a deranged first sound at the *apex* indicates *insufficiency* of the mitral valve, caused by vegetations or other result of endocarditis.

We have now disposed of derangements or abnormalities (which are always *morbid*) of the first sound of the heart both at the base and apex. They indicate nothing else but what we have said that they do.

Let us now proceed to pronounce upon derangements or abnormalities of the second sound (diastolic), should they be noticed either at base or apex: If the second sound is deranged, its greatest intensity or disturbance being at the *base* of the heart, it must necessarily indicate the exact opposite condition to that which we stated that derangements of the first sound indicated,—for exactly the reversed condition of affairs is taking place—the semilunar valves are shutting now, they were open then. The valves at the base are acting directly contrary to those at the point also; when one set is shut, the other set is open.* During the second sound we know that the aortic valves are closing, in order to keep the blood temporarily from flowing backward into the left ventricle (which is a reservoir of supply). So if there is a morbid second sound (diastolic) at the base, the valves of the aorta are *insufficient*. The front-gate has not closed tightly; there are vegetations—hardened *plaques* of fibrine, or bone, or cartilage which interfere with the integrity or pliancy of the delicate curtains which form this front flood-gate; and instead of the column of blood in the aorta remaining quiescent for a moment, as it should and does do in a state of health, some of it regurgitates into the dilating ventricle and gives a deranged, morbid second sound.

Therefore a morbid *second sound* at the *base* indicates *insufficiency* of the aortic valves.

* A third well-known relation may very properly be stated here to complete the sketch of these antagonisms and contrasts. This regards the cavities of the organ. The ventricles and auricles are synergetic only with themselves; when the former are contracting the latter are dilating, and *vice versa*.

Now suppose the deranged, morbid second sound has its seat of greatest intensity at the *apex*, instead of being at the base; it is very plain then that the back-gate, the mitral or bicuspid orifice, is narrowed, obstructed (stenosis), and the blood in passing through makes a noise. Because during the second sound (diastolic) the mitral orifice should be wide open to allow the blood from the auricle to enter noiselessly and fill up the ventricle, otherwise there would be no supply for the next systolic effort of the heart. If the orifice is obstructed or narrowed the blood does not pass through noiselessly as in a state of health—the second sound is abnormal; there is a murmur.

A disturbed second sound at the *apex* indicates then *stenosis* of the mitral orifice.

Our table now is very easily constructed, and being based upon eminently natural and scientific foundations, namely, the physical laws of the heart's structure, functions, and actions, it must serve as a ready method, enabling us, or anyone else—even the most uninstructed,—to make a diagnosis of all the uncomplicated organic diseases of the valves at the orifices of all the chambers of the heart. It is necessarily true and correct, and though it may seem very simple, it requires no thought to apply it to any case before us; nor is it necessary that we should at the time of applying it, understand *why* it is correct.

The formula and the order of the words to be recalled are:—

Stenosis.
Insufficiency.

Insufficiency.
Stenosis.

For example:—

At the Base.—A deranged first sound indicates *Stenosis* of the aortic, or pulmonary artery valves.

A deranged second sound indicates *Insufficiency* of the aortic, or pulmonary artery valves.

At the Apex.—A deranged first sound indicates *Insufficiency* of the bicuspid, or of the tricuspid valves.

A deranged second sound, indicates *Stenosis* of the bicuspid, or of the tricuspid valves.

All we have to do is to memorize those words in their order, as a formula, to elucidate at the bedside the valvular diseases of the heart. Observe what sounds are deranged at the base, then at the apex, and pronounce accordingly.

Of course the known relative positions of the four valves must guide us in deciding which of the two valves at the base or at the apex the abnormal murmur proceeds from, so as to distinguish between the valvular derangements of the right and left heart.

II. The above are what we consider to be the most essential facts with regard to the production and significance of the sounds and murmurs, heard upon auscultation of the heart, to be recognized and understood by everyone who examines this viscus.

Many will content themselves simply with the systolic and diastolic murmurs described above, and not extend their inquiries to the point where the diagnosis requires the consideration of the presystolic, post-systolic, intermediate murmurs, and those modified by the complications existing between the diseased valves, the chambers, the muscular structure and the related viscera.

But it is incumbent upon us to describe here some important murmurs long known to possess pathological signification, and indicating diseased conditions of great gravity. The characteristic features, the cause and explanation—the very existence even—of one or two others have been, and are being daily discussed by some of the ablest observers in our profession, including Skoda, Hoppe, Flint, Broadbent, Hayden, and others.

Dr. A. Flint lays special stress upon a "*mitral systolic non-regurgitant murmur.*" He contends that a murmur of the first sound, heard within a limited area *at the apex* (the mitral regurgitant only can be heard laterally and at the back,) may be due to roughness of the endocardial membrane without mitral incompetency, and, conse-

quently without a *mitral regurgitant current*. This is the murmur which is present in endocarditis.

We can also have murmurs on the right side of the heart indicating stenosis or insufficiency which—though not frequently met with—may coexist with other murmurs, namely: tricuspid direct, and regurgitant, (the latter not being very uncommon according to Broadbent,) and pulmonic direct, and regurgitant. These are generally consecutive upon other murmurs, result from secondary changes such as hypertrophy, dilatation, etc., and are distinguished, by their anatomical position, a venous or hepatic pulsation, dilatation of the right auricle and the cardiac end of the vena cava ascendens, the quality and pitch of the sounds, etc.—all which are explained with more or less clearness in the several treatises specially devoted to the subject, and to which we must refer the reader.

Mitral Stenosis.—We stated in the first part of this paper that a murmur at the apex, diastolic in time, was indicative of mitral stenosis. This is true, but it is necessary to add that, to speak more strictly, the diagnostic mark of this murmur is its occurrence at the *end of the diastolic period*, it is actually terminated by the systole of the heart, so that it is essentially a presystolic murmur.

The murmur is caused by the vibrations of the mitral curtains, especially when these curtains are united at their sides, leaving a narrowed orifice through which the mitral direct current of blood flows. It may occur without mitral lesion when there is free aortic regurgitation, because, as explained by Flint, there being blood in the ventricle, when the auricular contraction takes place the mitral curtains are floated out so as to be in contact with each other, and the mitral direct current passing between the curtains throws them into vibration precisely as when the orifice is narrowed. Mitral stenosis, to repeat, is produced by adhesions to each other of the mitral curtains, these forming a funnel-shaped space with the so-called button-hole opening—the curtains not having been made rigid by thickening or calcification. If these conditions be wanting, or if the force of the auricular contractions, as in advanced stages of the disease be wanting, when from dilatation the muscular force of the auricular contractions are weakened, the murmur disappears (Flint). So alcohol and digitalis may cause its return. Dr. Broadbent, in his recent article (*Am. J. Med. Sc.*, Jan., 1886) gives the following concise description of it, which we quote, as it is the most serious of the diseases of the valves, standing next to aortic regurgitation in order of gravity, and may be mistaken for mitral incompetency, which is the least so. According to Broadbent, children are especially liable to mitral stenosis, which gives rise to arterial embolism, great enlargement of the liver, and true hepatic pulsations. Before quoting what follows, we think it just to mention that Traube had long since called a "presystolic" murmur at the apex, a pathognomonic symptom of stenosis of the mitral valve; and Niemeyer had stated in his "Text-book" (7th ed., 1869) that "if the contracted orifice be not also roughened, if the stenosis be moderate, if the volume of the blood be reduced, there may be no sound;" . . . and "the second sound of the pulmonary artery is naturally considerably intensified."

"The pathognomonic sign of mitral stenosis is usually given as a presystolic murmur heard over a limited area to the inner side of the apex beat. It is not a smooth blowing murmur, but has a rough and vibratory character, and is often accompanied by a thrill perceptible to the hand at the same spot. Corroborative evidence is afforded by accentuation of the pulmonary second sound, the result of backward pressure in the pulmonary circulation, and, not infrequently, by want of synchronism in the closure of the pulmonary and aortic valves, giving rise to reduplication of the second sound. These are, in effect, the signs in the first stage (he makes three stages), but another important note must be added, viz., that the second sound is audible at and beyond the apex. With such a combination of signs the diagnosis is extremely easy; a murmur heard near the apex and followed by a first and second sound can only be presystolic. If further aid were wanted, it would be afforded by the character of the murmur, which, as has just been said, differs remarkably from other murmurs; it is not flowing and smooth, but vibratory, or in some instances rumbling. (Flint calls it vibratory and blubbering, and Niemeyer 'that slight whizzing sound,' or *frémissement cataire*—being also the most prolonged of the murmurs.) And again, the way in which it runs up to, and suddenly ends in the first sound, which tends to become short and loud, is highly characteristic."

The second stage is marked by the disappearance of the second sound at the apex and by the short, sharp character of the first sound, which also usually becomes very loud—an explanation of which is attempted by Dr. Broadbent. The third stage is marked by the disappearance of the presystolic murmur altogether. Persons die from this disease which would not be diagnosticated if the presystolic murmur were looked upon as a pathognomonic *sine quâ non*. The probable cause of the disappearance of the murmur, according to Broadbent, is the establishment of tricuspid regurgitation—the strain upon the pulmonary and tricuspid valves being very great. We must refer the reader to the article quoted, as well as to that of Dr. A. Flint in the same issue, as no more space is at our disposal.

Mitral Diastolic Murmur.—Professor A. Flint in his article (January issue, 1886, of the *Am. J. of the Med. Sciences*) refers to another mitral murmur, being unable to attribute to any one the credit of having first described it as an individual murmur. As regards its clinical recognition, he says, a murmur which follows the second sound, and ends before the contraction of the auricle, if aortic and pulmonary regurgitation be excluded, may be assumed to be a mitral diastolic murmur. It is easy to conceive of the mitral diastolic murmur being overlooked, if it be soft and feeble, when associated with a loud presystolic murmur, especially if the former be not sought after. It is produced by the current of blood from the auricle to the ventricle prior to the auricular contraction, and it is a mitral direct, but not a presystolic murmur. "In point of time it is diastolic, and the name mitral diastolic is an appropriate designation for it."

III. It was particularly necessary to avoid prolixity in the descriptions contained in the first division of this paper, which it was desirable to present with the greatest degree of clearness and precision; we will now add some particular suggestions pertinent to the subject, and a number of miscellaneous aphorisms which, being incontrovertible, will be of service as references to those who are investigating endocardial murmurs.

It must be observed that a murmur is not a natural, healthy heart sound; it is a fresh, *pathological* sound caused by alterations of an orifice or its valves by adventitious growths, contractions, etc.; hence eddies are set up in the blood, and a disturbed normal relation is established between the size of the orifices and that of the cavities of the heart.

Murmurs may or may not replace the normal heart sounds. Heart sounds, as stated by West,[2] may be modified, or even absent, without any murmur at all; and Professor Austin Flint asserts "that the quality of a murmur does not, in general, invest it with any special pathological or diagnostic symptom" ("Manual of Percussion," 1876, p. 213).

"No positive conclusions are to be drawn from the intensity of murmurs, their pitch or their quality" . . . "as a rule, murmurs which are weak, more than those which are loud, represent grave lesions" (*op. cit. sup.* p. 226).

Another practical fact to be noted is: that murmurs are transmitted in the direction in which the blood is passing at the time of their production. For example: systolic aortic murmurs are propagated upward, and diastolic aortic propagated downward toward the apex.

That a sound is systolic can readily be determined because it coincides with the impulse of the carotid, which, being quite near the heart, can be appreciated by the touch. It also coincides with the first sound of the heart and with the apex beat,—but the latter cannot be de-

pended upon, because it cannot always be heard. The radial pulse will not aid because it is not in accord with the first or systolic sound.

We had stated that there was a period of rest in the heart's action. This is not strictly true; yet varying intervals as to duration and rhythm do exist in the heart beats—the action of the organ being less violent and rapid during the presystolic period (when the ventricles are being filled from the auricles), *i.e.*, when the movement of diastole is approaching its end. This is known as the "long pause," and the organ then enjoys a certain repose. Dr. Austin Flint, Jr., gives this concise description of the diastolic period ("Physiol.," p. 51); (we italicize some lines): When the second sound occurs, the ventricles having become suddenly relaxed, the recoil of the arterial walls, acting upon the column of blood, immediately closes the semilunar valves upon the two sides. The auricles continue to dilate, and the ventricles are slowly receiving blood. *Immediately following the second sound*, during the first part of the interval, the auricles become fully dilated; and in the *last part of the interval, immediately preceding the first sound the auricles contract* and the ventricles are fully dilated. This completes a single revolution of the heart.

A reduplicated sound is often owing to a want of synchronism between two chambers of the heart—the valves of which ordinarily acting in concert give rise to a single sound. Irritability of the papillary muscles, and fatty degeneration give rise to tumultuous beating.

All tones arise from this "That a membrane passes from a state of less tension to a state of greater."

A distinct musical note is most uniformly associated with a narrowed orifice.

The terms stenosis, constriction, or obstruction are more properly applied to an orifice than to a valve; they imply that condition by which the effluent blood meets with abnormal resistance through contraction (from whatever cause) of the outlets of the heart; or again, the walls of the orifices may be thickened and contracted—the valves being sound; or there may be cohesion of the flaps; or vegetations, cartilaginous hardness and calcareous deposits may exist. Constriction and insufficiency may very naturally co-exist in any of the outlets of the heart—but more especially in the left chambers.

Every diastolic murmur alone authorizes the inference of structural changes in the valves, or pericardium; every systolic does not do so (Hoppe: On "Auscultation and Percussion").

The same authority says: You may have a systolic aortic murmur from inequalities on the surface of the valves which are turned toward the ventricle.

We must constantly remember the compensatory action of hypertrophy and dilatation of the ventricles and auricles, the slow process of these changes, and the stage which they have reached while estimating the character and signification of valvular murmurs. For example, a dilated or hypertrophied right heart, resulting from mitral stenosis, can propel so much blood into the pulmonary circuit that even the blood in the pulmonary veins, which are at the other end of the circuit, is subjected to heavy pressure. In consequence of this, to say nothing of the action of the auricle, the blood pours with such force and rapidity into the left ventricle as to completely neutralize the effect of the constriction of the mitral valve. In spite of the constriction, the ventricle receives blood enough, the aortic contents are not lessened, nor is the circulation retarded. In the same way, the fulness and tension of the pulmonary vein prevent any considerable regurgitation into the ventricle, notwithstanding the insufficiency of the valve—assuming that stenosis and insufficiency, as is often the case, coexist.

Where there is insufficiency of the aortic valves (sign: abnormal second sound, base), life may be prolonged for a considerable time; when, however, death ensues, it is sudden, resulting from cerebral apoplexy; where there is stenosis of the left auriculo-ventricular valve (sign: presystolic murmur at apex), life is continued for but a brief period—death, however, is not sudden as in the preceding case (Hoppe).

"Short breath is a symptom never missed in valvular disease of the mitral," as a consequence of hyperæmia of the lung which results from it; "hypertrophy corrects this in aortic disease" (Niemeyer).

Insufficiency of the aortic valve is more apt to be slowly followed by apoplexy and hyperæmia; constriction of the same reacts more on the lungs.

Valvular disease of the aorta gives rise to embolism with a frequence next to that of endo- and myo-carditis.

Strengthened apex impulse, the tortuous course of the smaller arteries, and the pulsations visible at the radial artery are signs of aortic insufficiency; but the *jerking pulse* is the only pathognomonic sign. In aortic stenosis the pulse is as small and incompressible, as it is hard and full in insufficiency.

In disease of the mitral the pulse is soft and irregular, and pulmonary complications are likely to arise.

Strengthening of the second tone of the aorta occurs in hypertrophy of the left ventricle when this is not caused by valvular defect; owing to abnormally increased obstruction in the peripheral part of the aortic system which opposes emptying of the left ventricle.

In endocarditis (complication with rheumatism in twenty per cent.), a blowing sound at the apex is not sufficient; you must wait for intensification of the second sound of the pulmonary artery to diagnose it. This last is caused by hypertrophy or dilatation of the right ventricle (a result of insufficiency of the mitral—developed in the majority of cases of endocarditis), and hence overloading of the pulmonary artery. The fuller the pulmonary artery becomes, so much the stronger does the shock grow which its semilunar valves must sustain during diastole.

In mitral insufficiency there is hypertrophy of all of the right heart; often dilatation of the auricles and ventricles—also of the pulmonary arteries and veins.

In mitral insufficiency and stenosis the pressure is upon the lungs, causing œdema and short breath; but patients often enjoy moderate health for a time. After a while the contents of the aorta diminish, the secretion of urine is lessened, the veins and capillaries become involved, the lips blue, the liver enlarged, with obstruction of the hepatic veins and bile; so gastric catarrhs ensue; the hæmorrhoidal, uterine and renal veins are obstructed, albumen appears and dropsy follows.

Niemeyer also refers, as follows, to the difficulty of diagnosing disease of the tricuspid:

In the extremely rare cases in which the right ventricle is the seat of endocarditis, similar symptoms may be made out at the lower part of the sternum, where we listen to the sounds of the tricuspid. It would be exceedingly difficult, however, to make a diagnosis here, as the right ventricle is hardly ever the sole seat of disease, and we should scarcely be able to distinguish whether the sounds were conducted from elsewhere or actually originated at the tricuspid.

Affections of the tricuspid valves are very usually connected with dilatation of the organ; but the dilatation or hypertrophy results from valvular disease—generally of the mitral.

Digitalis is useful in mitral insufficiency and "in many cases of cyanosis, dropsy, hepatic engorgement and suppression of urine caused by disease of mitral valves, because it diminishes or delays the necessity for the compensatory action of hypertrophy and dilatation."

In insufficiency of the tricuspid valves, (Bennett and Tanner question whether "disease of the tricuspid can be diagnosed,") blood regurgitates into the right auricle, causing turgescence and pulsation of the jugular veins, anasarca, congestion of the liver, dropsy and disease of the kidney. So these effects correspond with the above statement regarding the later stages of mitral stenosis and insufficiency. Tanner says tricuspid obstructive and pulmonary artery regurgitant are seldom if ever heard. Simple hypertrophy of the right heart is very rare.

F. Peyre Porcher.

[1] See Am. Jour. of the Med. Sciences for October, 1880.
[2] How to Examine the Chest. By Samuel West, M.D., Oxon. London, 1885.